POSTER
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A Novel Inositol Phosphate, Which Inhibits NPY-Induced Thyroidal Vasoconstriction, Fails To Block Acute Decreases in Thyroid Blood Flow induced by Sympathetic Nerve Stimulation. M. Dey, M. Michalkiewicz, L. Huffman and G.A. Hedge. Dept. of Physiology, West Virginia University Health Sciences Center North, Morgantown, WV 26506.

Sympathetic nerve fibers to the thyroid blood vessels contain both norepinephrine (NE) and neuropeptide Y (NPY). To assess the involvement of endogenous NPY in the sympathetic neural control of thyroid blood flow (TBF), a selective NPY antagonist, D-myo-inositol-1,2,6-triphosphate (PP56) was used in anesthetized rats. During all experiments, TBF was continuously monitored by laser Doppler blood flowmetry. Pretreatment for 5 min with PP56 (35mg/kg BW; iv bolus) blocked the decreases in TBF seen during exogenous NPY infusion (2.4 nmol/kg BW; iv; 4 min). However, this dose of PP56 had no effect on the thyroidal vasoconstriction induced by exogenous NE (15nmol;iv bolus). We next determined whether PP56 would affect the thyroidal vasoconstrictive response to stimulation of the cervical sympathetic trunks, which are the major sympathetic supply to the thyroid gland. Both the cervical sympathetic trunks were stimulated (30 Hz, 10V; 0.5msec; 2 min), 5 minutes after either saline or PP56 (35mg/kg BW) injection. Stimulation was repeated after 15 minutes. PP56 failed to block the vasoconstriction evoked by either the first or second stimulation of the cervical sympathetic trunks. Our results suggest that NPY is not involved during acute sympathetic vasoconstriction in the rat thyroid gland (Supported by NSF DCB-8904470).